valescence from any disease in which there has been great impairment of nutrition, such as typhoid fever, etc.

Second :— Hypertrophous cardiac dilatation.— In this form there is increase of the heart-cavities, accompanied by a slight increase of the thickness of the heart walls ; but the contractile power of the heart is diminished. This condition may occur as the result of a degeneration of eccentric hypertrophy, or it may occur independent of any hypertrophy, of the cardiac walls.

Third :—Atrophic cardiac dilatation.—In this form the capacity of the heart cavities is markedly increased, and the cardiac walls are markedly thinner than normal. Sometimes the ventricular walls diminish to not more than two or three lines in thickness, and the auricular walls may become so thinned that they will present the appearance of a simple membrane. Under these circumstances the contractile power of the heart is almost lost. Anatomically as well as clinically the significance of cardiac dilatation is in proportion to the excess of the capacity of the cavities over the thickness of the cardiac walls. A cardiac cavity may be very much increased in capacity, but so long as there is a corresponding increase in the muscular power of its walls sufficient to meet the demand of the increased work they are called upon to perform, there will be little or no disturbance to the general circulation. Eccentric hypertrophy and hypertrophous dilatation approach each other very closely, and it is often very difficult to draw the line of separation between them.

Morbid Anatomy.—One or all of the heart cavities may be the seat of dilatation. The shape of a heart when it has undergone dilatation is changed according to the cavity which is the seat of the dilatation. If the dilatation is confined to the right ventricle, the heart will be increased in breadth ; while if the dilatation affects mainly or only the left ventricle, the heart will be increased in length. Ordinarily when one cavity is dilated the remaining cavities are more or less affected in the same manner.

Cardiac dilatation occurs most frequently in the auricles ; next in the right ventricle ; and last of all in the left ventricle. While the left ventricle is less liable than the right to become the seat of dilatation it is more liable to become the seat of hypertrophy. When all the cavities are dilated, the entire organ is increased in size, and assumes rather an ovoid shape. When the ventricles are excessively dilated, the trabeculæ are sometimes reduced to the condition of fleshy tendinous cords. When the walls of the left ventricle are very much thinned, they collapse when the ventricle is opened. The anatomical changes which take place in the muscular tissue of the dilated cardiac walls vary with the degenerative process which precedes and attends the dilatation. When it results from pericarditis or myocarditis, there is serous infiltration and granular degeneration of the muscular fibres. When it is the result of fatty metamorphosis the muscular fibres undergo fatty degeneration, the process of which will be described under the head of fatty heart.

In hypertrophous dilatation, it is often impossible, even by microscopic examination to determine the exact changes which the muscular fibres undergo ; the abnormal state of the muscular fibres can only be determined by the other evidences of feeble heart power. You must be careful not to mistake a heart distended with blood and relaxed by putrefaction for a dilated heart. The distinctive marks of a heart softened by the putrefactive process are its extreme softness, its saturation with the coloring matter of the blood, and the evidences of decomposition in other parts of the body. Closely connected with the morbid anatomy of cardiac dilatation, is its causation.

Etiology. The causes of cardiac dilatation vary very widely. One class of causes may be included under the head of the immediate changes which take place in the muscular tissue of the walls of a heart that has undergone dilatation. I have already alluded to these. First, we have the changes in the muscular tissues which accompanies pericarditis and endocarditis ; second, fatty degeneration of the muscular fibres ; third, a cardiac dilatation which occurs with certain forms of protracted disease, such as typhoid fever, where the most careful microscopical examination will fail to detect any uniform change in the muscular fibre, except, perhaps a general atrophy of all the tissues. One or all these tissue changes may be regarded as causes of cardiac dilatation ; again all the causes of cardiac hypertrophy may become the causes of dilatation in a heart which has a feeble resistant power. This group of causes may be classed under three heads :—*First : internal pressure during a cardiac diastole.* The wall of a heart may become weakened by the changes which occur in certain prolonged diseases, or it may become the seat of serous infiltration or fatty degeneration ; then an abnormal pressure within its cavities during its diastole will cause the cardiac walls to yield beyond their normal limits. Such distension is certain to be followed by permanent dilatation of its cavities. Most of the valvular lesions which have recently occupied our attention may be the direct cause of such internal pressure during the cardiac diastole, after the manner I have already described in connection with the etiology of cardiac hypertrophy. Generally (as I have endeavoured to show you), when the cardiac cavities become distended beyond their normal limit, and thus temporarily lose their contractile power, rapid hypertrophy of the cardiac walls is developed, which compensates, and to a certain extent overcomes the dilatation. But if the cardiac walls are enfeebled by any of the degenerative changes to which I have referred, such compensating hypertrophy does not take place, and any valvular lesion which will permit a double current of blood to flow into a cardiac cavity during its diastole, the heart walls having become enfeebled by degenerative changes, will give rise to cardiac dilatation. *Second :* when the muscular tissue of the heart is the seat of primary fatty degeneration, after a time dilatation of the cavities takes place, the normal blood pressure being sufficient to produce it. In the same manner will a heart become dilated when its walls are the seat of myocarditis. That form of cardiac dilatation which follows typhus and typhoid fever or chlorosis, usually disap-